



PERSPECTIVE

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Heart Failure Pathophysiology and Symptoms

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Description

Heart failure is mostly caused by a decrease in the heart muscle's effectiveness as a result of injury or overload. Myocardial infarction, hypertension, and cardiac amyloidosis are just a few of the numerous disorders that might result in it. Changes to the heart's structure will occur as a result of these workload increases over time.

The heart of a person with heart failure may have a reduced force of contraction due to overloading of the ventricle. In a healthy heart, increased filling of the ventricle results in increased contraction force and thus a rise in cardiac output. Because the ventricles are overloaded, the heart of a person with heart failure may not contract as forcefully. A healthy heart's higher ventricular filling causes a rise in cardiac output since it increases the contraction force. This mechanism malfunctions in heart failure because the ventricle is overloaded with blood to the point that heart muscle contraction is ineffective. This is a result of the stretched out cardiac muscle's decreased capacity to cross-link actin and myosin filaments.

A failure of systole, diastole, or both may result in a decreased stroke volume. Reduced contractility is typically the reason why end systolic volume increases. Reduced ventricular filling, which happens as the ventricle's compliance declines, causes a decrease in end diastolic volume. In times of high oxygen demand, the amount of cardiac output that can increase is reduced as the heart works harder to fulfill normal metabolic demands.

As a result, heart failure patients often experience exercise intolerance. Loss of one's cardiac reserve, or the heart's capacity to contract more forcefully during demanding physical exercise, is what this translates

to. The heart is unable to satisfy the body's increased metabolic demands during exercise because it must use more effort to meet normal metabolic demands. An elevated heart rate, brought on by increased sympathetic activity to maintain a sufficient cardiac output, is a common finding in people with heart failure. This initially aids in heart failure compensation by maintaining blood pressure and perfusion, but it also puts additional strain on the myocardium and raises the need for coronary perfusion, which can exacerbate ischemic heart disease.

A potentially catastrophic aberrant cardiac rhythm can also be brought on by sympathetic activity. The muscle layer of the heart may physically get larger. The reason for this is that in an effort to increase contractility, the terminally differentiated heart muscle fibres enlarge in size. This can exacerbate the stiffness and lessen the body's capacity to relax during diastole. The ventricles may also grow, which adds to the enlarged size and spherical shape of the failing heart. Due to the heart's mechanical and ineffective contraction, the increased ventricular volume also results in a decrease in stroke volume.

Systolic dysfunction is a more obvious sign of heart failure. It can be summed up simply as a failure of the heart's pump function. The ejection fraction is reduced, which is its defining feature. Inadequate cardiac output results from attenuated ventricular contraction that cannot generate a sufficient stroke volume.

Typically, cardiac myocytes or their molecular constituents are the culprit, either through malfunction or destruction. Individual myocytes' molecular makeup is impacted by hereditary illnesses like Duchenne muscular dystrophy. Infiltration or inflammation can harm myocytes and the parts that make them up. Ox-

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idative stress and intracellular damage are caused by toxins and pharmaceuticals. The most frequent cause of injury is ischemia, which results in infarction and the creation of scars. Dead myocytes are replaced by scar tissue after a myocardial infarction, negatively reducing the myocardium's ability to contract. This can be seen on an echocardiography as aberrant or non-existent wall motion.

Heart failure brought on by diastolic dysfunction is frequently characterized by a stiffer ventricular wall

and is generally described as the ventricle's inability to relax properly in the backward direction. Pierre-Simon Laplace was the first to identify the ventricular walls' "stiffness" and contractility during diastole. This results in insufficient ventricular filling, which in turn affects the stroke volume (SV). SV is a mathematical concept that can be changed in many different ways. Elevated end-diastolic pressures are another side effect of ventricular relaxation failure, and the outcome is the same as in the case of systolic dysfunction.